opposed to auditory information in short-term memory. This is probably due to the fact that any visual information of this type is usually recoded into auditory information in the process of retention, so that this coding procedure imposes an additional strain on short-term retention. It would appear, from a still relatively small group of schizophrenic patients who have performed this test, that they show the same difficulty in the short-term retention of visual information which is so pronounced in older adults. Indeed, the pattern of response of the young schizophrenic patient on this test is comparable to that of a normal subject twice his age.

To summarize our investigations originated in a clinical study of young schizophrenic patients whose descriptions of their symptoms were systematically compared. From an analysis of this data, we derived the general hypothesis that many of the difficulties reported by these patients were secondary elaborations of a more basic disorder in the selective and inhibitory functions of attention. There would appear to be at least two factors operating in the disorder of selective attention as measured by our tests. The striking deterioration in the visual-motor performance of schizophrenic patients suggests that distracting auditory stimuli may directly affect the act of perception. The majority of our results, however, imply that the effects of distraction are chiefly exerted in the brief period between perception and response. It would seem that the relative inability of the schizophrenic patient to screen out irrelevant extraneous information results in an overloading of short-term memory. This interpretation would explain the poor performance of the schizophrenic patients on tasks which demand accurate perception and recall. To function effectively in a normal environment, the individual must, however, be able to process a much greater amount of information than that contained in the rather simple and artificial situation represented in our tests. Normally we are able to process at a more efficient rate by organizing the information into larger units, thus reducing the load on short-term memory. We do this automatically in our normal perception of speech by using the transitional bonds inherent in normal language structure and by screening out the redundant words which occur in most verbal communications. In the case of schizophrenia, one might suggest that the patient is unable to direct attention away from the redundant words and towards key words, so that these redundant words have a retroactive effect on the perception and recall of the passage as a whole. Thus the 'depatterning' effect demonstrated in the speech perception of schizophrenic patients may be viewed as part of the general disorder in selective attention and short-term memory. The indications that the disturbance in short-term retention is particularly pronounced in the case of visual information, might be explained by the suggestion in recent experimental studies (Conrad 1964) that visual information tends to be verbally encoded during perception. Once again, in the case of schizophrenic patients this seems to act as an additional strain on short-term memory, causing a further disturbance in retention. In this sense, the performance of schizophrenic patients is similar to that of normal adults of advanced years. Finally, we have seen that the disorders indicated by our test results are not evenly distributed within the schizophrenic group, but tend to be concentrated among hebephrenic patients.

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Psychiatric Symptoms and Parietal Disease: Differential Diagnosis

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In this country combined departments of psychiatry and neurology do not exist, and outside of Service medicine the term 'neuropsychiatrist' finds no place. None the less full and friendly communication occurs between our two specialties, to mutual advantage. Today psychiatrists are keenly alerted to the chance that organic brain disease might be responsible for some of the clinical pictures which, superficially at any rate, seem psychologically determined. Similarly we neurologists endeavour to bear in mind that pure psychological problems may masquerade as one of our organic cases.

With increasing attention to the alleged silent areas of the brain, the topic of differential diagnosis becomes all the more pertinent. The most forward regions of the frontal lobes, the parietal areas, and particularly that territory where post-parietal, superior temporal and anterior occipital cortices adjoin, constitute the highest evolved structures of the brain, with their syndromes of strange complexity, puzzling to the uninitiated.

Parietal disease with its bizarre and even flamboyant manifestations, so paradoxical, odd and

unpredictable, may be overlooked by the inexperienced in favour of some neurotic malady. The clinical problem is quite unlike the tidy and uniform consequences of spinal and peripheral disease.

Two disorders, common to psychiatric and neurological practice, may occasion difficulties in diagnosis or errors in assessment: I refer to hysteria and dementia.

There are many similarities between parietal symptoms and hysteria, and some of the difficulties in distinction will be dealt with later. Among the exasperating traits of a patient with parietal disease is a certain incommunicability, so that it may be difficult to secure his co-operation. Impersistence on his part may make it hard to hold his attention; or, having done so, to coax him to switch quickly from one task to the next. Hence the characteristic slowness of response, which is often extreme. Lastly, an inconsistency of performance may be observed whereby the patient may do one undertaking correctly but not the next. Or he may succeed in the task which a moment or so ago seemed to be beyond him.

Diagnosticians may therefore find it hard to decide whether a patient is an hysteric, or the victim of organic brain disease. Still more baffling are those patients who undoubtedly have a cerebral lesion but the clinical picture is distorted by an hysterical elaboration.

The other pitfall in diagnosis is dementia. Here the difficulty is not one of disentangling a psychogenic overlay, but of deciding between two organic pictures. How much of the problem is the result of a focal as opposed to a global lesion? Entities such as asphasia, imperception, apraxia and anosognosia are sometimes looked upon as fragments retrieved from the slag-heap of dementia. That may be so, but the assessment of individual cases is often anything but simple.

Let us go over some of the commonplaces of parietal disease emphasizing those syndromes which are reminiscent of a psychopathology.

Among the tactile aberrations of parietal disease there may occasionally occur some unexpected features such as hallucinations of touch, haptic perseveration and gross inaccuracy in the localization of stimuli. A patient may fancy that an object still lies in his hand, when actually it is empty. Or he may announce that he has been pricked with a pin. Or he may continue to perceive a contact long after the stimulus has been removed. Asked to name the site of stimulation, the patient may indicate a point far removed, or a mirror-opposite place on the other half of the anatomy, or even some region which is outside the body altogether.

In the domain of vision one may mention the phenomenon of object agnosia, so called, where an article is alleged to be seen but is not identified. With prosopagnosia, there is a specific inability to recognise well-known faces. Grave disorders of insight may occur, so that a hemianopia is not realized by the patient, and the fact of blindness may be unnoticed, or even denied. Strange metamorphopsias may occur, some of them so subtle as to constitute an organic de-realization or depersonalization. Hallucinations may be met with, ranging from crude photisms to elaborate phantasmagoria, including specular or heautoscopic illusions.

Disorders of corporeal awareness (body image) are among the better known parietal bizarrities. In the notorious anosognosia for hemiplegia the patient is unaware that anything is the matter with him; or he may even deny the fact that he is paralysed at all. Or he may grudgingly admit that the limb is a trifle heavy, and then go on to offer a gratuitous excuse which is wholly unconvincing. Thus he may put it down to rheumatism or a sprain. Still more pathological is the patient who stoutly denies the ownership of the affected arm and leg. Alongside such an attitude of rejection remarkable feats of confabulation may be found. The patient may assert that his powerless limb belongs to the man next door, or to his wife, or to a stranger who is in bed with him, or even to a corpse; or he may declare that a heavy piece of wood or a metal bar lies across his chest.

A woman of 39 with a left hemiplegia, hemianæsthesia and hemianopia, was garrulous and confused. She denied that she was paralysed, and she would have it that her left arm and leg belonged to her daughter Ann who had been sharing her bed for the past week. But in the next sentence she proclaimed that Ann was away in St Ives on a holiday. When the patient's wedding-ring was brought to her notice, she asserted that Ann had borrowed it to wear. The patient was encouraged to talk to Ann, and to tell her to move her arm. She then became confused and talked vaguely about Ann being asleep and not to be disturbed. Asked to indicate her own left arm and leg, the patient turned her head, and searched in a bemused way over her left shoulder.

Later the patient may regain full awareness of the fact that the limb is his, and that it is out of action, but he may adopt an odd proprietary or detached attitude towards it, as though it were a plaything or a pet. This is the phenomenon of 'personification'. Thus he may endow the limb with a nickname . . . 'George', 'Fanny', 'the lazy one', 'silly Billy', 'floppy Joe', 'Dolly Gray', 'old useless'; or merely speak of it as 'he' or 'she'. One of the most extreme instances of personification that I have seen was of a man who called his paretic left arm 'Lucky' or else 'the little monkey', and he would nurse it, pet it, stroke and caress it, tickle it, address it in baby

talk, and at meal-times even attempt to feed it with a spoon.

Hemiplegic patients may adopt various attitudes towards their disability. One patient may accept his defect in a light-hearted almost jocular fashion; this is what is called 'anosodiaphoria'. Or, by way of contrast, the patient may show a loathing or abhorrence of his disability, and he may speak of his paralysed member as being horrid, withered, shrunken, 'like a bird's claw', or 'the paw of a mummy'. He may cover it up out of sight. Or he may punch and pummel it. This is the phenomenon of 'misoplegia'.

The left arm of a patient with a right parietal lesion kept wandering about in the blind homonymous half field. When the patient wrote, the left hand would wander across and butt in and rest upon his right hand. Not recognizing this as his own he would exclaim 'Let go my hand!'. Later he would swear at his hand in exasperation: 'You bloody bastard! It's a lost soul, this bloody thing. It follows me around, and gets in the way when I read. I find my hand up here by my face, waving about.'

Somewhat less grotesque are the spatial disorders so typical of parietal disease. The patient may lose himself in the street, and even in the familiar environment of his own home. He may be unable to read a map, and to sketch one would be out of the question. Dressing, bathing, shaving, may be impossible, and a woman may be at a loss how to fix her hair or titivate her face. The drawings of such patients are often childish, even fantastic, especially when the task is to sketch some such intricately articulated object as a bicycle, a daisy, a clock-face, or an elephant.

An inability to carry out a motor skill is often found and even simple learned activities may be impaired. A housewife can no longer lay a table or cook a three-course dinner. A journey across the city necessitating train, tube and bus will be out of the question. Spatial defects, apraxia – constructional or otherwise – and disorders of corporeal awareness may combine so as to hinder and hamper performance. In one interesting psychomotor disorder a patient may perform an act and then undo it, or cancel it out, by means of a contrary action. He may put on his jacket and then take it off again: light a cigarette and stub it out. Mindful of the *Odyssey* this caprice is sometimes spoken of as the 'Penelope syndrome'.

What is the reason for this recurring psychiatric flavour in cases of parietal disease? Outfall of parietal function can only be part of the answer. A fundamental derangement in behaviour resulting from cerebral disorder irrespective of the site of the lesion must also play a part. For many years the conception of a cerebral *Grundstörung* has been mooted in neurology, though opinions have differed as to its nature.

In this connexion most attention has been directed towards the ideas promulgated by Goldstein, which though familiar, deserve a restatement. A brain-injured person undergoes a change in his total personality which is betrayed in the complexity of his behaviour. Some tasks are beyond his capacity, and unsuccessful attempts to perform them produce a distressful 'catastrophic reaction' which in itself further hampers efficiency. Consequently the patient's whole way of life becomes directed towards building up a harmonious milieu from which unattainable goals are excluded. In this way putative sources of catastrophic reaction are avoided, though not at a conscious level. As Goldstein put it: 'A defective organism achieves ordered behaviour only by a shrinkage of its environment in proportion to the defect.' This propitious milieu, or shrunken environment, is brought about in various ways. A press of activity; or a retraction into a state of relative inaccessibility; the adoption of substitute reactions; an excessive orderliness; these are some of the barricades erected by the organism to maintain composure. We now begin to understand some of the puzzling features of the patient's behaviour under examination, such as slowness of response; inconsistency of performance; elusive attention; ideational inertia; and persevatory phenomena. Performance will also depend upon the amount of 'energy' which is available; if one performance requires undue energy expenditure, some other performance will suffer. The patient's attitude to his own disability may be unexpected and, as a rule, the greater the loss of function the more complete the adjustment. And then pervading all activity – whether in the sphere of language or of motility – there is a drift away from the abstract, categorical or conceptual, towards the concrete.

Other philosophies of nervous function have also been invoked, and may illuminate the apparent obscurities. Von Monakow drew attention to the effects upon nervous activities of acute lesions, and he showed that the immediate effects are far in excess of what can be regarded as straightforward outfall phenomena. Over and above, there may occur widespread but temporary losses of function. To this shock effect the term 'diaschisis' is applied. Diaschistic effects are gradually lost as nervous equilibrium is slowly re-established uncovering the specific and permanent focal disabilities.

Yet another doctrine of dynamic neurology detracts somewhat from a strict localizationist attitude: I refer to the innate reluctance on the part of the organism to accept or admit the hard facts of a loss in function; but instead, to adopt attitudes whereby the disability is glossed over,

wholly or in part; or denied outright; or explained away by rationalizations and excuses, mainly of an extraneural sort. Some neurologists align these types of reaction with local parietal disease, but others believe that they are nonspecific, being manifestations of a more fundamental 'denial syndrome'. I must admit to a considerable sympathy with this latter view which serves to explain a lot of clinical data, which, because they do not fit in with preconceived ideas, are often brushed aside as inconvenient and unimportant. This in itself is a denial syndrome at the bedside. Here would belong those cases of Anton's syndrome associated with peripheral blindness, and not central; the so-called visual agnosias from lesions of the frontal lobes; the anosognosias after leucotomy, and many other nonconformist phenomena met with in neurological practice. Here too belong some of the 'negative cases' where there is much pathology but little disability.

One factor not to be neglected in our assessment of the effects of organic brain disease is the previous personality. The nature of the make-up of the individual may profoundly influence the subsequent reaction to crippling disease. One patient treats his hemiplegia with insouciance and flippancy, another reacts with depression and shame. We speak of the former as showing anosodiaphoria, the latter misoplegia. But the earlier personalities may well have been as different as the two modes of reaction. A preoccupation with physical efficiency and athleticism may lie behind the misoplegic who cannot support the trauma of a stroke; while the anosodiaphoric may have been a simple soul undismayed by his constitutional inadequacies.

That reaction depends upon premorbid personality, harks back to Hughlings Jackson, who promulgated the doctrine of 'the four factors of the insanities'. Jackson was not wholly consistent in his writings on this subject, and by 'insanities' he was referring to brain disease in a wide sense. His four factors were made up of (1) the size of the lesion; (2) the site of the lesion; (3) the abruptness of the lesion; and (4) the kind of brain that the lesion was affecting (Jackson 1874).

In the series of difficult cases described below parietal manifestations and psychiatric features were intertwined to an unusual degree. In passing I may say that with increasing experience, clinical assessment does not necessarily grow any easier in such cases. To the novice in neurology a particular case often appears as a clear-cut instance of say, parietal disease, or of dementia, or of hysteria. But I have found that the more knowledgeable the examiner, the less dogmatic he is. This dictum is borne out by the original and notorious case of Schneider studied in such detail by Goldstein & Gelb (1918). Many criticisms have subsequently been made, and though the patient is still alive, opinions still differ. The same remark applies to the so-called Wilbrand-Charcot syndrome of irreminiscence, which many consider to be hysterical.

Case 1

A married woman of 46 was first seen fifteen years ago. Following a thrombophlebitis she developed a sudden left-sided hemianopia, presumably embolic in nature. She did not recognize her half-vision, but proclaimed that everything at first seemed muzzy. Back home after her stay in hospital, she said that the flat did not look the same. Out of doors she felt uncanny and unreal: people looked different. Everything and everybody appeared unattractive – older, uglier, untidy. Whirring sensations, like a spring, made her continuously support her head with her hand. She could not bear to gaze at any loudly patterned material: the view from her window was different and unpleasing. To look at a bunch of flowers or a bowl of fruit gave her a feeling of fear though a single bloom was tolerable. She was uncertain about the topography indoors and out, and in the hospital ward when she went to the sluice she would place her blue cardigan on the bed to serve as a marker.

Her introspection and depression have continued to date, that is, for thirteen years, though latterly she has become better adjusted. Here then is a right parieto-temporal lesion precipitating a complex neurotic illness coloured by elaborate visual complaints. Never before had there been any psychiatric breakdown, but the previous personality was of a masterful, tomboyish individualist with a rigid obsessional personality, having been regarded by her colleagues in the office as a very 'tough egg'.

Case 2

A man of 84 had developed, eighteen months before, a right-sided hemiparesis. Gradually the affected side became the seat of intolerable dysæsthesiæ. The paretic hand became occupied by feelings of heat, coupled with intense pins and needles. To get relief he would constantly wiggle his fingers, or rub the hand, or tuck it under his armpit. As the patient said . . . 'the hand is awful and it worries me. It won't relax. . . . All day it wants to be doing something. The tingling makes it fidget. I try to push the tingling away with my other hand, as though I were putting on a glove.' The patient was completely preoccupied by his symptoms and could talk of nothing else.

Again this was essentially an organic problem, the lesion being in the left thalamo-parietal peduncle. The clinical picture was quite unlike the usual syndrome of unilateral pain and hyperpathia, but took the form of an intractable dysæsthesia with hypochondria and depression, organically determined.

Case 3

A man aged 52 was known to have had a mild stroke causing a transient hemiparesis and aphasia. Fifteen months later he consulted us because when he re-

turned to his research work in atomic physics, he found he could no longer assemble pieces of apparatus. Nor could he now comprehend Greek mathematical symbols. He could not make use of a logarithm table. Later it came to light that the patient had for long been misleading his doctors, his wife, and many others too, by falsely claiming to be a highly-qualified scientist, decorated for his wartime services. Actually he was a humble laboratory technician.

He subsequently developed mild angina, and later he turned up at my clinic having suddenly lost his sight two weeks before. His behaviour was theatrical. In great distress he proclaimed he could not see, but on closer testing he began to pick out items here and there in his environment. First it was a name on a bed-board far across the ward. Then it was my neck-tie; then the head of a pin in my lapel. But faces, forms, and objects were not perceived. Soon afterwards it became obvious that there existed a dense and persistent left homonymous hemianopia. The patient died shortly after from a cardiac infarction.

In this case we see the effects of two cerebral accidents occurring in a mythopœic psychopath. The onset of hemianopia was complicated temporarily by a total blindness, the nature of which is conjectural. Even now I find it hard to decide in retrospect whether I was witnessing an hysterical overlay, or the phenomenon of diaschisis.

Case 4

An arteriopath of 50 was admitted with a recent complete blindness of abrupt onset. Asked whether there was anything wrong with his vision he replied that it had not been too good of late. 'Can you see me?' 'Yes.' 'What am I wearing?' 'I can't tell you . . . I can't see things distinctly.' 'Can you see to read?' 'Yes.'

Ten days later the following conversation took place: 'Do you complain of anything?' 'No.' 'Why then are you in hospital?' 'I'd have to enquire further.' 'What stops you going back to work now?' 'Nothing.' 'Could you manage your intricate work as a dental mechanic?' 'Yes.' 'Could you ride your bike to work?' 'Yes.' 'I'm holding something up in front of you; what is it?' 'I can't see.' 'How do you explain that you can't see this?' 'I don't know.' 'Is your eyesight all right?' 'It's not too good.' 'Are the lights on or off?' 'Off.' 'If they were switched on would you be able to see?' 'Yes.' 'Can you see this?' (a pair of scissors). 'No.' 'Take hold of it.' 'They're scissors!' 'Now that they are in your hand, look at them. Can you see them now?' 'Yes.'

This is an example of Anton's syndrome and there is no reason to implicate any neurotic overlay. The state of the patient's mind was, however, interesting. At first he did not realize that he was blind. Later he developed some insight, but of a partial and inconsistent kind. From day to day the state of awareness varied, and indeed it would fluctuate from moment to moment during a single interview. He always minimized his disability, tending to project it. At all times he was completely unperturbed and uncomplaining. Often he rationalized, or confabulated. Whenever he identified an object by touch, he would thereafter proclaim that when he held the object up to his eyes, he could see it. This last-named phenomenon has not before been explicitly described in the literature.

Case 5

In many respects this case is the reverse of the others. A hairdresser of 52 was examined in hospital where it was noticed that some of his clothes were inside out, and that he had difficulty in dressing. There were also signs suspicious of a constructional apraxia. But the patient's own story was of a very different kind. For three years he had been a victim of a compulsion to write down whatever people were heard to say, car numbers, placards, notices, and even the dates on the coins in his pocket. In his barber's shop he would have to dart into the WC in order to make a note of what his last customer had said, together with his replies. Eventually his job became untenable. He filled a series of notebooks with his frantic scrawls. At home he would read, and re-read, and index these records, even though the contents of the subject-matter was quite trivial.

The problem here was surely a psychiatric one, and the parietal signs which had been unearthed were probably irrelevant. Perhaps they were the product of overzealous medical probing. Incidentally the patient eventually lost his graphic compulsions and then showed no clinical anomaly at all.

The next three cases represent perplexing problems where, despite the most thorough study, diagnosis remained in doubt throughout the years.

Case 6

A boy of 14 had been laid up for years in early childhood on account of Pink disease. Thereafter his eyesight was regarded as very poor, and he grew up dependent upon his mother for almost every care. Visual testing revealed his acuity to be 6/6 in each eye, but the fields of vision were grossly restricted in a 'tubular' or 'tunnel' fashion. The patient was a very timid dullard. Many physicians and eye specialists had seen him and opinions oscillated between hysteria and organic disease. Air studies were later carried out and revealed small porencephalic cysts involving the post-parietal and occipital regions on both sides.

The last finding lent considerable support to those who favoured an organic pathology. Whether the problem was entirely explained along these lines remains a matter of opinion.

Case 7

A woman, now aged 47, has been under neurological scrutiny for fourteen years. She first came to hospital with an eight-month history of headache, followed by fainting attacks; later by double vision; and, three weeks before admission, by a loss of feeling down the left half of the whole body. She has remained in that state ever since, showing a severe one-sided paralysis,

anæsthesia, and visual loss. The reflexes, however, were unaffected, and such ancillary tests as the EEG, angiography, and air studies were negative. When examined three years after the trouble began, she said, looking at her left arm, 'I can't realize it's my own. I can't feel it at all. It could be someone else's. It might just as well be amputated. It's just as though I were cut in half. One side of my face seems loaded with cocaine.'

The nature of this case was hotly debated. It may or may not be significant that while most of the senior staff at Queen Square regarded the problem as an organic one, the house officers in the privacy of their quarters were in confident agreement that the patient was an hysteric. Perhaps the truth was vouchsafed to the physicians of the intermediate grade, when they spoke of the patient as an hysterical psychopath afflicted with some obscure organic lesion of the right parietal lobe. The problem is still unsolved.

Case 8

Over ten years ago a man of 25 was admitted to the National Hospital with a four-year history of 'attacks', and of misty vision. Because of this he could not read, for print looked foreign to him. Nor could he comprehend pictures or identify faces. As he gazed at the wallpaper in his room, a piece of the pattern seemed to dislodge itself and shoot out of the window. Eight years later he was readmitted. He could not follow television if there were more than two persons on the screen. He could identify objects separately but not in apposition. People were still meaningless, and their faces all looked like eggs.

Seen again a month ago he was working as a school caretaker. He still could not identify objects except after prolonged deliberation, and pictures could not be interpreted.

No abnormal physical signs were ever found in this patient, and all investigations were negative. His 'attacks' were possibly epileptic though not typical. In this case too, opinions were divided as between psychogenic and organic disease, though most neurologists who interviewed him believed that there was a parieto-occipital lesion beneath a superstructure of hysteria.

Lastly, we may consider the distinctions, if any, between parietal signs and dementia. Here again, neurologists of experience and ability, and psychiatrists, are apt to differ in their evaluation of a given case. To one, the clinical picture may represent a straightforward dementia, hence implying no focal lesion. To another, the same syndrome is interpreted as a combination of apraxia, aphasia and agnosia, and may therefore possess practical repercussions as regards localization. Once again, overconfident opinions often indicate in practice a relative inexperience.

As regards distinguishing features, let me quote some of the criteria which I ventured to lay down some years ago, as being more suggestive of a global dementia:

(1) An inability to plan ahead; to anticipate events; or to undertake novel enterprises, while still capable of coping with routine matters.
(2) Conduct which is incongruous, or socially offensive. Incontinence in circumstances which are blatantly inappropriate. Sexual offences; indecencies; miserliness, or else financial extravagance; uninhibited speech or behaviour.
(3) An affective state which is inappropriate, especially when there is also an impaired control over the expression of the emotions, including an unbridled irritability.
(4) A disorientation which is global.
(5) Ignorance of, or indifference to, current affairs.
(6) A conspicuous and global defect of memory.
(7) Progressive poverty of speech, with an inability to reel off a list of objects belonging to a class. To this may be added odd verbal mannerisms and iterations.
(8) The appearance of such neurological anachronisms as bilateral forced grasping and groping; sucking movements; lip and snout reflexes; pathological laughter and crying; and a tendency to stuff objects into the mouth.
(9) Neurological signs which are symmetrical and bilateral, such as for example, bilateral extensor plantar responses.

The foregoing are merely guides, and one must not be misled into oversimplification. It may be objected that the mental concomitants of diffuse diseases differ merely in degree from those of focal lesions. May not the algebraic summation of a number of isolated lesions be tantamount to a dementia? Take a case of multiple secondary tumours of the brain. The first metastasis may be followed by, say, a Gerstmann's syndrome. Dementia in the strict sense of the word cannot be said to exist. Soon other focal symptoms develop, like a dyslexia. Still the patient is not demented, *sensu strictu*. Shortly afterwards he becomes drowsy, apathetic and taciturn, though capable of being roused to converse sensibly. Still later he is bedfast and has to be fed. He is now incontinent. Some confusion exists as to time and place; his memory is failing; his attention is difficult to hold. So by gradual stages the patient has lapsed into a dementia, the product of half a dozen metastases seen at autopsy scattered throughout the brain. Few would be bold enough to assert at which point in the clinical course the cerebral outfall symptoms constituted a veritable dementia. It could be argued that right from the start, the first focal manifestation was potentially productive of a dementia. In other words, a Gerstmann syndrome, an apraxia, a visual imperception may each of them represent in itself a *petite démence;* a putative, subclinical dementia. Expressed differently it might perhaps be claimed that to speak of 'dementia' or 'absence of dementia' is unwise, for in reality there is but a clinical spectrum which

represents the reaction of the organism to cerebral modification, ranging by easy stages from success to failure. I must confess that this holistic mode of thinking is one which I find the most satisfying.

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As a neurosurgeon on the staff of a psychiatric hospital I have had referred to me from time to time some interesting cases of parietal lobe disorder with a psychiatric flavour. Naturally, these have been mostly tumour cases. The modes of presentation have been very variable, but I would like to exclude consideration of cases which can occur in either hemisphere where there is a marked hemiparesis, both sensory and motor. Also, I would like to exclude cases with various degrees and forms of dysphasia or of Gerstmann's syndrome with finger agnosia and acalculia such as can occur with lesions of the dominant temporal lobe. These cases are obvious and easy diagnostic problems. Instead I shall confine myself particularly to disorders of the right or minor parietal lobe associated with more subtle disorders such as anosognosia, constructional apraxia, or epilepsy associated with spatial disorientation or of panoramic memory.

The terminology used to describe these disorders is voluminous and, consequently, often confusing. I shall try to keep it simple. Agnosia is a failure to recognize familiar objects presented to the senses of vision, hearing, or of touch. Apraxia is a difficulty in synthesizing and carrying out common motor or mental actions. The right parietal lobe can give rise to many agnosic syndromes, but the most striking are the body-scheme agnosias and, in particular, anosognosia, where the patient seems to deny that anything is wrong with the left or affected side of his body and ignores it. This is often associated with a constructional apraxia. Let me instance a case:

Case History

A 55-year-old business man

Three months before his admission, while in the street, he felt somebody touch his left shoulder, but on looking round he could see no one. This was repeated on three or four occasions. One month before his admission his wife noticed clumsiness of the left arm; he would knock objects off the table with it. Shortly afterwards, he had a number of accounts to tabulate. Try as he could he did not manage to arrange them in proper order, but when his secretary arranged them for him he added them up correctly. This was dismissed as due to fatigue from overwork, and he took a few days off in bed at home to rest. From his bed he would telephone his subordinates and astound them with feats of memory concerning past business deals. Then one day, to prove himself well, he got up, drove his car round the block and on returning to the garage crashed the left front wing against the garage door. This led to his referral.

On examination he kept protesting how well he was. However, it was obvious that he was ignoring his left side. His signs included a left-sided attention hemianopia, marked left-sided sensory impairment and a constructional apraxia. He could walk, read, write and identify his fingers and those of the examiner, but he could not copy easily and correctly simple designs made with three matches. When he sketched he failed to complete the left-hand side of his drawings and did not notice the omission. There was no motor weakness, and no papilloedema. Arteriography and needle biopsy established that he had a right parietal glioma, which was inoperable.

Localization is not always what it seems. A tumour in the posterior part of the right frontal lobe can occasionally produce much the same syndrome.

Epilepsy involving the right parietal lobe has also interesting features. In addition to sensory phenomena referred to the left side of the body, the patient during seizures may have illusions concerning objects on his left-hand side. Thus, one patient, during seizures, would describe the right-hand side of the faces of people opposite her as being distorted and horrible, or that sometimes people on the left seemed to be walking in slow motion. I have also had two patients with right parietal lesions who developed panoramic memories in their seizures.

This symposium stresses differential diagnosis. In examining such patients I would make a plea for careful examination of the visual fields to see whether there are any defects in attention in the left homonymous fields, to test for constructional apraxia, to ask the patient to sketch common objects to see whether details are ignored on the left-hand side, and to test the sensory functions of the left side of the body, particularly for stereodiagnosis, two-point discrimination and postural recognition in the fingers. Ultimately, neuroradiological studies such as carotid arteriography or lumbar encephalography may be required. In this way the diagnosis in most patients with these interesting phenomena can be established.